Vital Organ Perfusion During Assisted Circulation by Manipulation of Intrathoracic Pressure

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Background. We have previously shown, in dogs with severe cardiac depression, that modest cyclic increases in intrathoracic pressure, starting synchronously with left ventricular isovolumic contraction, significantly increase aortic flow and pressure. However, little is known of changes in vital organ perfusion during this technique of assisted circulation.

Methods and Results. We studied regional organ flow using radioactive labeled microspheres in 13 20–25-kg mongrel dogs. In the control group, after chemical induction of cardiac depression with verapamil and propranolol, coronary flow fell from 129.1±14.4 to 51.6±11.3 ml/100 g/min (p<0.005) and continued to decline over a 14-minute time period (flow was 32.2±11.5 ml/100 g/min at 7 minutes and 20.7±9.5 ml/100 g/min at 14 minutes [n=6]; all p<0.05). In the intervention group, regional blood flow was evaluated before and after the induction of cardiac depression and also during assisted circulation using 400-msec, 20–25-mm Hg intrathoracic pressure increases delivered by a circumthoracic pneumatic vest, starting synchronously with left ventricular isovolumic contraction. In the intervention group, coronary flow fell from 119±26.7 to 47.9±13.1 ml/100 g/min 1 minute after the induction of cardiac depression (p<0.005). With the initiation of assisted circulation, coronary flow increased to 55.8±19.2 ml/100 g/min at 7 minutes and fell to 23.1±15.9 ml/100 g/min on termination of assisted circulation at 14 minutes (p<0.05 and p=NS versus control group flows at 1 and 14 minutes, respectively). During assisted circulation, cerebral, renal, and small intestinal flows also increased (all p<0.05 versus flows during myocardial depression). No significant increase in hepatic flow was observed.

Conclusions. In the canine model, manipulation of intrathoracic pressure appears to be an effective, short-term, noninvasive means of not only increasing aortic pressure but also increasing vital organ perfusion during cardiogenic shock. Further studies are needed to assess the usefulness of this technique of assisted circulation in humans. (Circulation 1991;84:279–286)

Heart failure remains a major cause of mortality and morbidity. Changing trends in angioplasty, bypass surgery, and cardiac transplantation have increased the therapeutic op-

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gators\textsuperscript{3-5} have studied noninvasive external counterpulsation. External compression to the legs and abdomen as a means of noninvasive assisted circulation was tried in 1974 and was shown to increase cardiac index after myocardial infarction.\textsuperscript{3} It did not gain widespread use, however, because of associated soft tissue injury, the lack of benefit in patients with severe peripheral vascular disease, and the cumbersome nature of the technique itself.\textsuperscript{3-5} Positive pressure ventilation was also evaluated as a technique to augment cardiac output.\textsuperscript{6-8} The use of cyclic intrathoracic pressure variations synchronized to the cardiac cycle as a noninvasive means of circulatory support was first proposed in 1981\textsuperscript{9} and was shown to significantly increase carotid flow and pressure. Pinsky and Summer\textsuperscript{10} augmented cardiac output in intubated patients with heart failure by applying nonsynchronized prolonged pressure waves to the chest and abdomen. Pinsky et al\textsuperscript{12} extended these observations in 1985 and reported increased left ventricular (LV) stroke volume in dogs with \(\beta\)-blocker–induced LV depression when cyclic increases in thoracic pressure occurred during early systole. However, this study and others that preceded it did not evaluate vital organ flow.

The development of a pneumatic vest system that could increase intrathoracic pressure without trauma or the need for simultaneous ventilation has allowed us to study in depth the potential for circulatory support by manipulation of intrathoracic pressure.\textsuperscript{13} The effectiveness of this system has been demonstrated in animals and in humans with cardiac arrest.\textsuperscript{14,15} In addition, using this system in dogs with chemically induced cardiac depression, Beyar et al\textsuperscript{16} demonstrated that modest cyclic increases (25 mm Hg) in intrathoracic pressure, lasting 400 msec and starting synchronously with LV isovolumic contraction, increase aortic flow and pressure and simultaneously decrease estimated LV transmural pressure.\textsuperscript{16} The clinical potential of this technique appears to be considerable, but little is known of vital organ perfusion during such manipulation in intrathoracic pressure. Decreases in transmural pressure could facilitate coronary flow, and the increase in aortic pressure could augment cerebral flow during assisted circulation. On the other hand, it is possible that the increases in intrathoracic pressure with this technique could decrease effective cerebral perfusion by raising intracranial pressure\textsuperscript{17} and decrease coronary perfusion by raising right atrial pressure. Beyar et al\textsuperscript{18} report only a modest increase in aortic diastolic pressure during assisted circulation by manipulation of intrathoracic pressure. This increase, coupled with the increase in right atrial pressure during the systolic rise in intrathoracic pressure, could reduce coronary perfusion. This study was designed to determine vital organ perfusion during noninvasive assisted circulation by manipulation of intrathoracic pressure. Regional blood flow was measured (using radioactive labeled microspheres) during assisted circulation with intrathoracic pressure wave parameters that had been previously defined as being optimal for augmenting aortic flow and pressure.\textsuperscript{16}

**Methods**

Two protocols were implemented after approval from the Johns Hopkins Medical Institutions Animal Care and Use Committee. These protocols conformed to the guiding principles of the American Physiological Society. In protocol 1, changes in coronary flow and hemodynamics during prolonged cardiac depression were assessed (control group). In protocol 2, the effect of assisted circulation by manipulation of intrathoracic pressure on vital organ perfusion was studied (intervention group).

**General Animal Preparation**

Thirteen mongrel dogs (20–25 kg) were anesthetized with phenobarbital (4 mg/kg i.v.) and intubated. All dogs were ventilated with a Harvard ventilator (model 607, Harvard Apparatus, South Natick, Mass.) with a tidal volume of 10 ml/kg at a rate of 10–15 breaths/min. Supplemental phenobarbital was administered as needed to maintain anesthesia. A left-sided thoracotomy was performed, and the ascending aorta, just above the coronary ostia, was exposed. For dogs in protocol 2, an aortic flow probe (model 501A, Carolina Medical Electronics, King, N.C.) was positioned and secured around the exposed ascending aorta. The flow probe had been calibrated with blood before and after the initiation of these studies. In all dogs, a small pericardial incision was made, and bipolar atrial and ventricular electrodes were sewn to the left atrial appendage and LV apex, respectively. The chest was closed in layers and evacuated of air, and the flow probe wires and electrodes were tunneled under the skin and brought out near the lower rib cage. Micromanometer-tipped catheters (Millar, Houston) were inserted in the femoral arteries and veins and advanced to the LV, thoracic aorta, and right atrium. Changes in right atrial pressure were used to estimate changes in intrathoracic pressure.\textsuperscript{16} In addition, both axillary arteries were exposed and cannulated with thin, polyethylene catheters that were advanced and positioned in the brachiocephalic arteries. These catheters were subsequently used for arterial blood sampling.

Data were recorded on an eight-channel recorder (model 2800S, Gould, Inc., Cleveland, Ohio) and were also digitized at a rate of 100 Hz and stored by computer on a disk. After recording baseline aortic flow and aortic, LV, and right atrial pressures, cardiac contractility was depressed with high doses of intravenous propranolol (23±12 mg) and verapamil (23±13 mg), and the heart was sequentially paced atrioventriculatly after bradycardia (<60 beats/min) developed. The heart was paced at a rate of 72 beats/min with an atrioventricular pacing interval of 100 msec using an R wave coupled-pulse generator (model 5837, Medtronic Inc., Minneapolis, Minn.). In all dogs verapamil and propranolol were administered until aortic flow decreased by 40% or greater.
and peak LV pressure was reduced below 100 mm Hg. Two to 3 minutes was allowed to confirm the hemodynamic effects of propranolol and verapamil, and the protocol was then initiated.

**Microsphere-Determined Blood Flow Measurements**

Baseline blood flow and blood flow following cardiac depression and during assisted circulation were determined by injecting 15±3-μm-diameter radiolabeled microspheres (141Ce, 113Sn, 103Ru, 95Nb, 153Gd, 46Sc, New England Nuclear, Boston) into the LV. Regional blood flows were determined according to techniques previously described.18,19 Reference samples were withdrawn from the two axillary artery catheters with a dual syringe pump (model 600, Harvard Apparatus). Withdrawal was started 15 seconds before injection and continued for 3 minutes at a rate of 3.8 ml/min for samples drawn before the induction of cardiac depression and 1.9 ml/min for 6 minutes for all samples drawn after cardiac depression had been induced and also during assisted circulation. The validity of using microspheres for low-flow states has been previously documented.18,20

**Circulatory Assistance System**

The system used to generate intrathoracic pressure variations has been described in detail elsewhere.13 In brief, this system consisted of an inflatable vest connected to a pneumatic system controlled by solenoid valves and fed by an external source of compressed air. A dedicated computer controlled the timing and duration of intrathoracic pressure waves relative to the atrial signal from the atioventricular sequential pacemaker. The amplitude of the intrathoracic pressure wave was set by controlling the vest inflation pressure. Vest inflation pressure was titrated to get the desired change in intrathoracic pressure. The intrathoracic pressure wave parameters used in this study had been previously defined as being optimal for augmenting aortic flow and pressure.16 During this study, the rise in intrathoracic pressure was synchronous with LV isovolumic contraction, lasted 400 msec, and was 20–25 mm Hg in magnitude.

**Experimental Protocol**

**Protocol 1: Time course of coronary flow during prolonged cardiac depression.** The purpose of this phase of the study was to evaluate coronary blood flow over time in this model of pharmacological cardiac depression. Six dogs were studied. The first microsphere injection was made before the induction of cardiac depression to establish baseline organ flow. Once a stable cardiac depression level had been obtained (time zero), three sequential microsphere injections were made at 1, 7, and 14 minutes with 6-minute blood sample withdrawals after each injection. This group of dogs served as the control group and defined the hemodynamic characteristics of the model used.

**Protocol 2: Assessment of regional flow during assisted circulation.** The purpose of this study was to compare regional flows during cardiac depression to flows during assisted circulation by manipulation of intrathoracic pressure. Seven dogs were studied and served as the intervention group. The timing of microsphere injections was the same as noted above. However, in this protocol, on completion of blood sample withdrawal after microsphere injection at 1 minute of cardiac depression, assisted circulation was initiated at 7 minutes and continued for an additional 6 minutes. The second microsphere injection was made during assisted circulation. The final microsphere injection was made at 14 minutes, on termination of assisted circulation. Tissues were harvested, and tissue and blood sample counts and flow were determined as previously described.17–19

Hemodynamic data were analyzed by a program that averaged 5-second samples and calculated the average aortic flow and the average peak systolic and diastolic pressure (relative to atmosphere) for each measurement location during the data-sampling period. Computer pressure readings were compared with visual pressure readings from the strip-chart recording to confirm the accuracy of the measurement. Estimated systolic LV transmural pressure (defined as peak LV systolic pressure minus peak change in right atrial pressure, assuming that right atrial pressure reflected the change in intrathoracic pressure) was also calculated.

**Statistical Analysis**

Values of pressures and flows were obtained from 5-second averages. Differences in paired data and group data were tested using a two-tailed paired t test or an analysis of covariance, as appropriate. Data are expressed as mean±SEM.

**Results**

**Hemodynamics and Coronary Flow During Prolonged Cardiac Depression: Control Group**

Table 1 and Figure 1 depict the changes in hemodynamics and coronary flow for a 14-minute period after the induction of pharmacological cardiac depression. These experiments also established the characteristics of the animal model used. At 1 minute after the onset of cardiac depression, a significant decrease in aortic and LV systolic pressures and a significant increase in LV diastolic and right atrial pressure were observed. At 7 and 14 minutes of cardiac depression, a gradual decrease in both aortic and LV systolic and aortic diastolic pressures was noted but did not achieve statistical significance. Coronary flow fell from 129.1±14.4 to 51.6±11.3 ml/100 g/min (p<0.005) after the induction of cardiac depression at 1 minute and continued to decrease for the first 7 minutes (p<0.005), likely reflecting ongoing drug effect. At 14 minutes, coronary flow was still lower (20.7±9.5 ml/100 g/min). The ongoing fall in coronary flow during sustained cardiac depression was likely due to changes in coronary vascular resistance as a consequence of increased
(unopposed) \(\alpha\)-adrenergic activity after \(\beta\)-blockade. To test the efficacy of assisted circulation, a separate group of dogs was studied.

Hemodynamics and Regional Flows During Assisted Circulation: Intervention Group

During protocol 2, as depicted in Figure 1, coronary flow was comparable with the flow in the control dogs before the induction of cardiac depression and at 1 minute after the induction of cardiac depression. During assisted circulation at 7 minutes, significantly greater coronary flow was noted in the intervention group than in the control group (55.8±19.2 versus 32.2±11.5 ml/100 g/min, \(p<0.05\)). After termination of assisted circulation, coronary flow fell, and there was no difference in coronary flow between the two groups of dogs at 14 minutes (\(p=NS\)). During assisted circulation, a significant increase in blood flow to the brain, kidney, and small intestine was also noted (Table 2). No significant change in hepatic flow was observed. On termination of assisted circulation, all organ flows fell significantly.

Figure 2 depicts the changes in hemodynamics during assisted circulation in a representative dog. Table 2 lists hemodynamic and regional flow data for four time periods: before the induction of cardiac depression (control), 1 minute after the onset of cardiac depression (before intervention, 1-minute depression), during assisted circulation (intervention, 7-minute depression), and after the termination of the intervention (after intervention, 14-minute depression). The increase in aortic and coronary flow during assisted circulation (at 7 minutes) was accompanied by a simultaneous increase in aortic and LV pressures and a significant decrease in estimated LV transmural pressure.

Discussion

The mechanism for blood flow during cardiac arrest in humans remains unclear but is likely due to both manipulation of intrathoracic pressure and cardiac/vascular compression. Manipulation of intrathoracic pressure has, however, been shown to be a potent mechanism for generating blood flow during

### Table 1. Hemodynamics During Sustained Cardiac Depression (Protocol 1)

<table>
<thead>
<tr>
<th>Pressure (mm Hg)</th>
<th>Control</th>
<th>1 minute</th>
<th>7 minutes</th>
<th>14 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>116.3±7.09</td>
<td>74.7±4.08*</td>
<td>74.5±7.2</td>
<td>61.5±10.4</td>
</tr>
<tr>
<td>Diastolic</td>
<td>85.3±6.69</td>
<td>51.3±4.6*</td>
<td>55.5±6.1</td>
<td>44.8±9.7</td>
</tr>
<tr>
<td>LV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>130.5±5.9</td>
<td>74.3±3.7*</td>
<td>70.8±6.9</td>
<td>62.75±9.2</td>
</tr>
<tr>
<td>Diastolic</td>
<td>6.3±2.8</td>
<td>23.6±4.8†</td>
<td>17.5±1.9</td>
<td>16.5±1.6</td>
</tr>
<tr>
<td>Mean RA</td>
<td>7.3±1.5</td>
<td>21.5±2.6*</td>
<td>21.0±3.4</td>
<td>21.75±2.6</td>
</tr>
<tr>
<td>Coronary flow (ml/100 g/min)</td>
<td>129.1±14.4</td>
<td>51.6±11.3*</td>
<td>32.2±11.5*</td>
<td>20.7±9.5†</td>
</tr>
</tbody>
</table>

Values are mean±SEM. LV, left ventricular; RA, right atrial.
*\(p<0.005\) and †\(p<0.01\) vs. corresponding value in preceding column.

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**Figure 1.** Bar graph showing microsphere-determined blood flow during cardiac depression for 14 minutes. Coronary blood flow was measured in six dogs (control group) before (PRE DEP) and at 1, 7, and 14 minutes after induction of cardiac depression. Coronary and brain blood flows were measured in seven dogs (intervention group) before and at 1, 7, and 14 minutes after induction of cardiac depression. However, in the intervention group, assisted circulation (by manipulation of intrathoracic pressure) was initiated at 7 minutes and continued for an additional 6 minutes; the final measurement was determined after termination of assisted circulation (at 14 minutes). During assisted circulation at 7 minutes, there is a significant increase in coronary flow in the intervention group vs. the control group. During assisted circulation, brain blood flow also increases.
TABLE 2. Hemodynamics and Vital Organ Flows During Cardiac Depression and Assisted Circulation (Protocol 2)

<table>
<thead>
<tr>
<th>Cardiac depression</th>
<th>Control</th>
<th>1 minute (Before intervention)</th>
<th>7 minutes (Intervention)</th>
<th>14 minutes (After intervention)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic flow (ml/min)</td>
<td>1,821.0±88.7</td>
<td>589.0±96.0*</td>
<td>728.0±119.0†</td>
<td>337.9±79.4†</td>
</tr>
<tr>
<td>Pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>126.6±6.6</td>
<td>81.9±4.3*</td>
<td>102.7±5.9*</td>
<td>54.7±8.5*</td>
</tr>
<tr>
<td>Diastolic</td>
<td>96.9±8.3</td>
<td>57.9±3.5*</td>
<td>58.0±3.9</td>
<td>33.4±6.2*</td>
</tr>
<tr>
<td>LV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>134.4±7.9</td>
<td>78.6±4.5*</td>
<td>102.1±5.5*</td>
<td>52.9±8.7*</td>
</tr>
<tr>
<td>Diastolic</td>
<td>3.1±0.7</td>
<td>13.4±2.0*</td>
<td>15.4±1.5</td>
<td>13.0±1.5</td>
</tr>
<tr>
<td>Mean RA</td>
<td>6.1±1.3</td>
<td>19.4±1.3*</td>
<td>27.9±1.8</td>
<td>18.7±0.9</td>
</tr>
<tr>
<td>LV transmural</td>
<td>133.6±8.1</td>
<td>78.5±6.0*</td>
<td>69.3±7.3†</td>
<td>49.9±8.9*</td>
</tr>
<tr>
<td>Organ flow (ml/100 g/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary</td>
<td>119.0±26.7</td>
<td>47.9±13.1*</td>
<td>55.8±19.2</td>
<td>23.1±15.9*</td>
</tr>
<tr>
<td>Brain</td>
<td>24.9±2.1</td>
<td>21.0±1.7‡</td>
<td>36.0±5.0†</td>
<td>12.0±4.0†</td>
</tr>
<tr>
<td>Kidney</td>
<td>341.9±45.6</td>
<td>128.7±37.4*</td>
<td>155.4±42.2‡</td>
<td>89.3±69.3*</td>
</tr>
<tr>
<td>Small intestine</td>
<td>44.0±6.4</td>
<td>14.6±4.1†</td>
<td>22.9±8.2*</td>
<td>8.1±4.1*</td>
</tr>
<tr>
<td>Liver</td>
<td>7.6±3.6</td>
<td>3.3±1.8*</td>
<td>2.7±0.8</td>
<td>1.9±1.5</td>
</tr>
</tbody>
</table>

Values are mean±SEM. LV, left ventricular; RA, right atrial. *p<0.005, †p<0.01, and ‡p<0.05 vs. corresponding value in preceding column.

cardiac arrest in humans and animals.14,15,22 The hemodynamic effectiveness of intrathoracic pressure manipulation is likely determined to a significant degree by the techniques used to produce the increases in pressure, with maximal augmentation of flow and pressure being reported with the use of specialized pneumatic vest systems.14,23 The development of such a system that can increase intrathoracic pressure with minimal trauma has allowed us to extend these initial favorable observations during cardiac arrest and pursue the potential for circulatory support by manipulation of intrathoracic pres-

FIGURE 2. Recordings from a representative dog in the intervention group showing hemodynamics during assisted circulation, with the rise in intrathoracic pressure being synchronous with the QRS. When synchronized vest inflation and assisted circulation cease, a fall in aortic flow, aortic pressure, and left ventricular (LV) pressure is noted. ECG, electrocardiogram; RA, right atrial.
sure in the nonarrested but severely depressed circulation. Using this pneumatic vest system, Beyar et al. showed that significant aortic flow and pressure augmentation occurred when the rise in intrathoracic pressure was synchronous with, preceded, or followed LV isovolumic contraction by 40 msec. This increase in aortic flow was associated with an increase in aortic pressure and a simultaneous decrease in estimated LV transmural pressure as confirmed by these experiments. These data also suggested that relatively low-pressure intrathoracic waves synchronous with LV contraction could provide an effective noninvasive circulatory-assist method for the failing heart in the dog and likely had potential for human use. However, the clinical applicability of any technique of assisted circulation would depend largely on its effect on vital organ perfusion. Our present studies confirm that assisted circulation by manipulation of intrathoracic pressure also increases vital organ flow. The magnitude of increase in coronary flow is similar if not greater than that reported with the intra-aortic balloon pump.

These data are encouraging, since the relatively low range of intrathoracic pressures used during assisted circulation (25–30 mm Hg) can be achieved in humans with chest pressures that are physically tolerable and may not necessitate endotracheal intubation. Though the dogs in these studies were intubated, preliminary studies from our laboratory indicate that cyclic increases in chest pressure without endotracheal intubation can effectively increase intrathoracic pressure. The chest pressure used in this study to produce intrathoracic pressure waves of 25–30 mm Hg was approximately 100–250 mm Hg. Newer vest designs transmit pressure more effectively and allow a lower chest inflation pressure to be used. Cyclic chest pressure increases to 100–140 mm Hg have been tested in several normal volunteers and have been found to be tolerable. This feature increases the clinical potential of this technique of assisted circulation and similarly increases its versatility. The rise in intrathoracic pressure during this technique of assisted circulation is likely due to the fact that the sudden rise in pressure during chest inflation results in transient airway collapse, which reverses with chest deflation. This technique of noninvasive assisted circulation is additionally attractive in that it also increases infradiaphragmatic organ and peripheral flow, thus overcoming some of the limitations of the intra-aortic balloon pump. In comparison with the intra-aortic balloon pump, this technique of assisted circulation has several potential advantages including improved infradiaphragmatic organ flow, its noninvasive nature, and relative ease of application and use.

The mechanism for the increase in coronary flow as noted during assisted circulation is likely related to the fall in estimated LV transmural pressure and decreased LV afterload. The increase in aortic diastolic pressure was minimal, and no significant increase in coronary perfusion pressure was found during assisted circulation. The increase in coronary flow likely results from changes in vascular resistance (which may, in turn, relate to changes in LV end-diastolic volume) and possibly from increased systolic coronary flow. Phasic coronary flow was not measured in these studies, since the primary goal was to measure vital organ and regional perfusion, which is best achieved by microsphere techniques. A circumferential coronary flow probe to measure phasic flow could have been inaccurate at the low perfusion pressures seen in these experiments, and other cannulating flow probes would require extensive surgery that would likely distort mediastinal anatomy and consequent pressure transmission to vascular chambers. The higher cerebral flow noted during assisted circulation by manipulation of intrathoracic pressure is easily explained by the increase in aortic, and likely carotid, arterial pressure.

In this study, other markers of myocardial function or metabolism were not assessed. However, the reduction in LV end-diastolic volume and LV transmural pressure would favor a reduction in myocardial oxygen consumption. This study is also limited in that long-term side effects of manipulation of intrathoracic pressure were not directly assessed. However, in animals with severe heart failure, reproducible changes in hemodynamics can be achieved by repeated periods of circulatory support (by manipulation of intrathoracic pressure) for up to 10–20 minutes with no evidence of tachyphylaxis. At the end of each experiment in this study, the dog’s chest and abdomen were surgically opened, and no evidence of gross lung, rib, or visceral injury was found. It is possible that after prolonged periods of intrathoracic pressure manipulation, greater visceral trauma may be encountered. The chest pressure used in this study was approximately 100–250 mm Hg. This is lower than that used by Halperin et al., who reported no evidence of visceral or bony trauma after sustained intrathoracic pressure manipulation for more than 20 minutes with nearly double this chest pressure. Therefore, it is less likely that lower chest pressures would result in trauma, even after prolonged use.

Another limitation of this study is that blood gases were not monitored during the protocol. Since the mechanism of intrathoracic pressure rise is transient airway collapse, hypoxemia may have occurred during assisted circulation. This was not formally evaluated. However, there was no clinical evidence of hypoxemia, though the possibility of a modest degree of hypoxemia cannot be ruled out. The other limitation of this study is that, as demonstrated by the control dogs, the animal model of “cardiac depression” used is inherently unstable and represents an “acute” model of heart failure. Though vascular pressures declined over 20 minutes, the similarity of hemodynamics at 1 minute and 14 minutes after depression in the control and
assisted-circulation dogs allows us to study the hemodynamic effects of assisted circulation before, during, and after circulatory support. Since hemodynamics at 14 minutes are comparable in the two groups of dogs (control and intervention), the fall in aortic pressure in the intervention group after circulatory support cannot be a consequence of the intervention. This technique of assisted circulation has only been studied in an acute model of heart failure; based on the mechanism of benefit, it is likely to be effective in situations of chronic severe heart failure as well.

Thus, it appears that modest increments (25–30 mm Hg) in intrathoracic pressure occurring synchronously with isovolumic LV contraction and lasting for 35–50% of the cardiac cycle can effectively increase peripheral and vital organ flow and pressure without increasing estimated LV transmural pressure. The technique used to manipulate intrathoracic pressure is unique in that it is noninvasive and is effective at low vent pressures. With this noninvasive technique of assisted circulation, significant flow and pressure augmentation can be realized. Though these initial results are encouraging, they must be viewed with caution, since the duration of intervention studied was short and the magnitude of flow increase was modest. Significantly longer durations of support should be studied, and the effectiveness of this technique in terms of sustained hemodynamic benefit and safety after such prolonged support should be established. If found to be effective, this technique could potentially be used during patient transportation or immediately after cardiopulmonary resuscitation. By virtue of its noninvasive character, this technique of assisted circulation may lend itself to a wide spectrum of clinical uses.

References

Key Words • circulation, noninvasive assisted • blood flow • intrathoracic pressure
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